How accurate is a CT scan in identifying acute strokes?

The short answer - noncontrast cranial computed tomography (CT) is terrible at identifying acute stroke in the first hours after symptom onset.[1,2] Nevertheless, it is the initial imaging method of choice for patients with acute onset of neurologic deficits, because it is readily available in most emergency departments, can be performed rapidly, and is the best method to identify acute hemorrhage or other mimics of acute stroke.[1] In essence, in the first hours after onset of a neurologic deficit, we perform computed tomography to look for everything but ischemic stroke.

The classic hypodensity changes of ischemic infarction may not be visible on a computed tomographic scan until more than 24 hours after the onset of symptoms.[3] Subtle signs of infarction visible in the first few hours after an ischemic stroke include cerebral edema, mass effect (sulcal effacement, ventricle distortion), and loss of the gray-white matter junction.[4,5] In some cases, a hyperdense middle cerebral artery sign, indicative of the thrombus that has induced the stroke, may be visible on computed tomographic images obtained during the first few hours after symptom onset.[4,5] These signs, although helpful when present, typically are not seen on the initial scan, and their absence in no way rules out ischemic stroke.

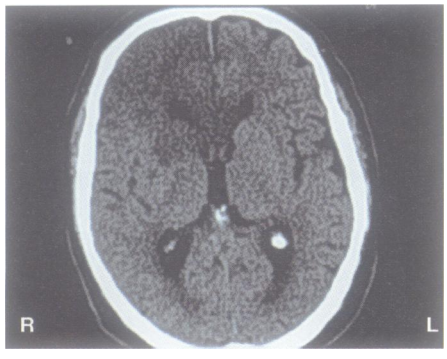

Figure 1 Noncontrast computed tomographic scan in a patient with new onset neurologic deficits shows a hypodensity in the right frontal area. The size and degree of attenuation of this image suggests a large infarct, or an infarct in the subacute (2-21 days) time period.

From 10% to 15% of strokes are hemorrhagic.[1] The majority of this type of stroke are visible on the computed tomographic scan, but it necessitates finding a competent reader to appreciate the finding. A study involving a convenience sample of emergency medicine, general radiology, and neurology physicians revealed that each group had a sensitivity for identifying hemorrhage of 73%, 87%, and 87%, respectively.[6] Thus, not all physicians can reliably identify intracerebral hemorrhage on computed tomographic scans and then safely select candidates for thrombolytic therapy.

Although the signs of ischemic infarction often are not detectable using computed tomography in the first few hours after symptom onset, findings on these initial scans indicative of a large infarct in the territory of the middle cerebral artery must be identified as they represent relative contraindications to thrombolytic therapy.[7-9] Results indicate that a convenience sample of emergency medicine, general radiology, and neurology physicians identified 67% to 93% of large ischemic infarcts.[10] Although experienced neuroradiologists can identify hemorrhage, studies suggest that subtle signs of ischemic infarction may not be identified as reliably. Experienced neuroradiologists achieve complete agreement (interobserver reliability) for the identification of large ischemic infarcts in the territory of the middle cerebral artery (more than one third of the middle cerebral artery territory) on initial computed tomographic scans in 52% to 82% of patients.[4,5,10]

Many other neuroimaging methods are potentially helpful in the evaluation of acute stroke, but magnetic resonance imaging (MRI) is the most common and informative adjunct to computed tomography. Standard T1- and T2 – weighted magnetic resonance imaging sequences identify acute ischemia more predictably and at an earlier time interval from symptom onset than does computed tomographic scanning.[2] Newer magnetic resonance imaging techniques, including diffusion-weighted imaging and perfusion imaging, appear to be even more sensitive and specific in the first few hours after symptom onset than are standard magnetic resonance images.[11] Diffusion-weighted imaging helps clinicians identify 98% of infarcts within a few hours of the onset of ischemia in human studies and is effective at differentiating old from new ischemic infarctions.[12,13] Combining diffusion-weighted imaging with perfusion imaging may provide prognostic information as it identifies both ischemic and irreversibly damaged brain tissues.[12]

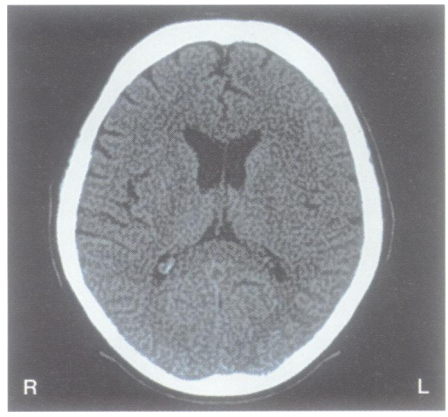

Figure 2 Noncontrast computed tomographic scan in another patient with new onset neurologic deficits reveals a left middle cerebral artery territory ischemic infarction. Note the subtle changes including sulcal effacement and loss of the normal gray-white matter junction of the left parietal lobe.

The major downside to magnetic resonance imaging when compared with computed tomographic scanning is that it is more time consuming. For ischemic stroke interventions, such as the administration of tissue plasminogen activator, for which the window of opportunity to begin treatment is 3 hours, it is essential to identify intracranial hemorrhage to avoid morbidity. Computed tomographic scanning is still considered the best means to identify acute hemorrhage, although some authors suggest special magnetic resonance images may be as accurate.[14] In addition, magnetic resonance

imaging is not readily available around the clock at many hospitals, the images are more susceptible to movement artifact, and some patients are precluded from this type of study (eg, because they are too large for the scanner, they have intracranial surgical clips or a pacemaker).

In conclusion, cranial computed tomographic scanning often does not identify early acute ischemic infarction, even when scans are interpreted by expert readers. This method is, however, the best means to rule out acute bleeding and other stroke mimics. As more specific therapies for cerebral ischemia become available, magnetic resonance imaging (diffusion-weighted imaging and perfusion imaging) and magnetic resonance angiography appear promising both for identifying acute strokes and for selecting among interventions.

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RESEARCH TO MAKE YOU THINK

Genetics Determines Your Pain Threshold

Anecdotal evidence has shown that people vary greatly in their sensitivity to pain and in their response to analgesics. Now, new research indicates that there is a genetic basis for differences in pain perception. The findings may lead to a more tailored approach towards determining doses of analgesic and to a better understanding of opiate addiction.

Working with mouse models, scientists from Johns Hopkins University in Baltimore and the National Institute of Drug Abuse identified a candidate gene involved in the regulation of nociception (pain perception) (Proceedings of the National Academy of Sciences USA 1999;14:7752-7755). The gene encodes the mu opiate receptor (mor), the primary target of morphine and other opiates.

The researchers, led by George Uhl of Johns Hopkins University, found that pain perception in mice, as measured by length of time for a response to temperature and pressure stimuli, varied inversely in relation to the density of the mu opiate receptors displayed. The more opiate receptors available, the smaller the response to the stimulus.

Mice that lacked mu opiate receptors had a lower threshold for reacting to pain: they responded to stimuli that were 66% of the strength of the stimulus required to elicit a response in mice with normal mor densities. For mice with half the normal number of receptors, pain reactions occurred when the strength of stimulus was about 80%.

Using eight different strains of mice, through genomic analysis, the researchers then traced the differences in mor density to polymorphisms in the regulatory regions of the mu opiate receptor genes. Several candidate promoter DNA sequences were identified. The promoter is a region of DNA lying upstream of the actual gene to which proteins bind that "promote" the transcription and ultimately the expression of the gene—in this case the opiate receptor. Sequence comparison of the gene encoding the mu opiate receptor showed that it is highly similar between mice and men, suggesting that similar regulatory differences may account for differences in pain perception in humans.

Interestingly, although studies of frontal cortex positron emission tomography in humans have shown that the density of mu opiate receptor varies by up to 50% in individuals, no convincing differences in the ability of receptors to bind with opiates has been shown, so the number of receptors alone may determine pain sensitivity.

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